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<i>DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>			
<input type="checkbox"/>	L1	oncolytic and NDV	46

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Search Results - Record(s) 1 through 6 of 6 returned.

1. Document ID: US 20060216310 A1

L4: Entry 1 of 6

File: DWPI

Sep 28, 2006

DERWENT-ACC-NO: 2006-754038

DERWENT-WEEK: 200677

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TITLE: Treatment of cancer in mammal having tumor, e.g. colon adenocarcinoma, cervical carcinoma, or melanoma, by administering intravenously more than one dose of composition comprising live Newcastle Disease Virus

INVENTOR: LORENCE, R M; REICHARD, K W

PRIORITY-DATA: 1994US-0260536 (June 16, 1994), 1993US-0055519 (April 30, 1993),  
2006US-0441201 (May 26, 2006)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>US 20060216310 A1</u>	September 28, 2006		025	A61K039/12

INT-CL (IPC): A61K 39/12; C12Q 1/70

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#) [Claims](#) [KMC](#) [Drawn D](#)

2. Document ID: US 7056689 B1

L4: Entry 2 of 6

File: DWPI

Jun 6, 2006

DERWENT-ACC-NO: 2006-400487

DERWENT-WEEK: 200677

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TITLE: Treating cancer in mammal having a tumor, comprising intravenously administering multiple doses of a composition comprising live purified Newcastle disease virus to a mammal

INVENTOR: LORENCE, R M; REICHARD, K W

PRIORITY-DATA: 1994US-0260536 (June 16, 1994), 1993US-0055519 (April 30, 1993)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>US 7056689 B1</u>	June 6, 2006		027	G01N033/574

INT-CL (IPC): G01N 33/574

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMIC	Drawn D.
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3. Document ID: AU 2003287472 A8, WO 2004043222 A2, US 20040131595 A1, AU 2003287472 A1, EP 1578451 A2, JP 2006510741 W

L4: Entry 3 of 6

File: DWPI

Nov 3, 2005

DERWENT-ACC-NO: 2004-420027

DERWENT-WEEK: 200629

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TITLE: Use of a negative-stranded RNA virus for treating a mammalian subject having a carcinoid tumor, and for decreasing symptoms of carcinoid syndrome, such as diarrhea, flushing or fatigue

INVENTOR: LORENCE, R M; MAJOR, P

PRIORITY-DATA: 2003US-457034P (March 24, 2003), 2002US-423952P (November 5, 2002), 2003US-0700143 (November 3, 2003)

## PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>AU 2003287472 A8</u>	November 3, 2005		000	A61K048/00
<u>WO 2004043222 A2</u>	May 27, 2004	E	017	A61B000/00
<u>US 20040131595 A1</u>	July 8, 2004		000	A61K048/00
<u>AU 2003287472 A1</u>	June 3, 2004		000	A61B000/00
<u>EP 1578451 A2</u>	September 28, 2005	E	000	A61K048/00
<u>JP 2006510741 W</u>	March 30, 2006		015	A61K035/66

INT-CL (IPC): A61B 0/00; A61K 35/66; A61K 35/76; A61K 38/00; A61K 39/155; A61K 39/17; A61K 48/00; A61P 1/00; A61P 1/12; A61P 17/00; A61P 35/00

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMIC	Drawn D.
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4. Document ID: AU 2005201079 A1, WO 200062735 A2, AU 200042469 A, US 20030165465 A1, JP 2003530301 W, HU 200302278 A2, EP 1390046 A2, CN 1477964 A, MX 2001010393 A1

L4: Entry 4 of 6

File: DWPI

Apr 7, 2005

DERWENT-ACC-NO: 2000-656408

DERWENT-WEEK: 200533

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TITLE: Treating neoplasms including cancer and solid tumors in a mammal comprises administering interferon-sensitive, replication-competent clonal RNA or DNA viruses such as paramyxovirus and herpesvirus

INVENTOR: BON BORSTEL, R W; GROENE, W S ; LORENCE, R M ; RABIN, H ; ROBERTS, M S ; VON BORSTEL, R W ; LORANCE, R M

PRIORITY-DATA: 1999US-0292376 (April 15, 1999), 1997US-0948244 (October 9, 1997), 1998US-0168883 (October 9, 1998), 2002US-0167652 (June 13, 2002), 2005AU-0201079 (March 10, 2005)

## PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>AU 2005201079 A1</u>	April 7, 2005		000	A61K039/00
<u>WO 200062735 A2</u>	October 26, 2000	E	108	A61K000/00
<u>AU 200042469 A</u>	November 2, 2000		000	A61K000/00
<u>US 20030165465 A1</u>	September 4, 2003		000	A61K048/00
<u>JP 2003530301 W</u>	October 14, 2003		134	A61K035/76
<u>HU 200302278 A2</u>	October 28, 2003		000	A61K038/17
<u>EP 1390046 A2</u>	February 25, 2004	E	000	A61K031/70
<u>CN 1477964 A</u>	February 25, 2004		000	A61K031/70
<u>MX 2001010393 A1</u>	April 1, 2004		000	A61K000/00000

INT-CL (IPC): A01N 63/00; A01N 65/00; A61K 0/00; A61K 0/00000; A61K 31/282; A61K 31/573; A61K 31/70; A61K 35/76; A61K 38/00; A61K 38/17; A61K 39/00; A61K 45/00; A61K 48/00; A61P 35/00; A61P 35/02; A61P 35/04; A61P 43/00; C07J 5/00; C12N 7/00; C12N 15/86

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5. Document ID: AU 2002325406 B2, WO 9918799 A1, AU 9896038 A, EP 1032269 A1, HU 200003911 A2, CN 1281336 A, JP 2001519175 W, NZ 503664 A, MX 2000003467 A1, AU 2002325406 A1, AU 2005237176 A1

L4: Entry 5 of 6

File: DWPI

Dec 8, 2005

DERWENT-ACC-NO: 1999-277360

DERWENT-WEEK: 200654

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TITLE: Treating tumours by infection with interferon-sensitive viruses, - effective against large tumours that do not respond to chemotherapy

INVENTOR: GROENE, W S; LORENCE, R M; RABIN, H.; ROBERTS, M S; VON BORSTEL, R W

PRIORITY-DATA: 1997US-0948244 (October 9, 1997), 2002AU-0325406 (December 24, 2002), 2005AU-0237176 (November 25, 2005)

## PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>AU 2002325406 B2</u>	December 8, 2005		000	A01N063/00
<u>WO 9918799 A1</u>	April 22, 1999	E	094	A01N063/00
<u>AU 9896038 A</u>	May 3, 1999		000	A01N063/00
<u>EP 1032269 A1</u>	September 6, 2000	E	000	A01N063/00
<u>HU 200003911 A2</u>	February 28, 2001		000	A01N063/00
<u>CN 1281336 A</u>	January 24, 2001		000	A01N063/00
<u>JP 2001519175 W</u>	October 23, 2001		104	C12N007/04
<u>NZ 503664 A</u>	August 30, 2002		000	C12N007/02

<u>MX 2000003467 A1</u>	October 1, 2001	000	A01N063/00
<u>AU 2002325406 A1</u>	April 3, 2003	000	A01N063/00
<u>AU 2005237176 A1</u>	December 15, 2005	000	A01N063/00

INT-CL (IPC): A01N 63/00; A61K 38/21; A61K 48/00; A61P 35/00; C12N 7/02; C12N 7/04; C12N 15/01; G01N 33/15; G01N 33/50

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6. Document ID: WO 9425627 A1, AU 9468213 A, EP 696326 A1, JP 09503995 W, AU 699487 B, EP 696326 B1, EP 1314431 A2, DE 69432409 E, ES 2196026 T3, EP 1486211 A1, JP 2005187484 A

L4: Entry 6 of 6

File: DWPI

Nov 10, 1994

DERWENT-ACC-NO: 1994-358299

DERWENT-WEEK: 200677

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TITLE: Treatment of cancer with Newcastle Disease Virus - opt. in combination with another anti-cancer agent

INVENTOR: LORENCE, R M; REICHARD, K W

PRIORITY-DATA: 1993US-0055519 (April 30, 1993)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
<u>WO 9425627 A1</u>	November 10, 1994	E	044	C12Q001/70
<u>AU 9468213 A</u>	November 21, 1994		000	C12Q001/70
<u>EP 696326 A1</u>	February 14, 1996	E	000	C12Q001/70
<u>JP 09503995 W</u>	April 22, 1997		038	A61K035/76
<u>AU 699487 B</u>	December 3, 1998		000	C12Q001/70
<u>EP 696326 B1</u>	April 2, 2003	E	000	C12Q001/70
<u>EP 1314431 A2</u>	May 28, 2003	E	000	A61K035/76
<u>DE 69432409 E</u>	May 8, 2003		000	C12Q001/70
<u>ES 2196026 T3</u>	December 16, 2003		000	C12Q001/70
<u>EP 1486211 A1</u>	December 15, 2004	E	000	A61K035/76
<u>JP 2005187484 A</u>	July 14, 2005		028	A61K039/17

INT-CL (IPC): A01N 63/00; A61K 35/76; A61K 37/66; A61K 39/12; A61K 39/17; A61K 39/42; A61K 49/00; A61P 35/00; C12N 7/00; C12Q 1/70; G01N 33/574

[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#) | [Claims](#) | [KWMC](#) | [Drawn D.](#)

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Terms	Documents
L3 and NDV	6

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NEWS 20 JAN 16 IPC version 2007.01 thesaurus available on STN  
NEWS 21 JAN 16 WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data  
NEWS 22 JAN 22 CA/CAplus updated with revised CAS roles  
NEWS 23 JAN 22 CA/CAplus enhanced with patent applications from India  
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NEWS 25 JAN 29 CAS Registry Number crossover limit increased to 300,000 in multiple databases

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NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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=> oncolytic

1228 ONCOLYTIC  
68 ONCOLYTICS

L1 1289 ONCOLYTIC  
(ONCOLYTIC OR ONCOLYTICS)

=> NDV

1420 NDV  
12 NDVS  
1422 NDV  
(NDV OR NDVS)

=> L1 and L2

16 T1 AND T2

=> D L3 IBIB ABS 1-16

L3 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2007:18747 CAPLUS

**TITLE:** In vivo efficacy of systemic tumor targeting of a viral RNA vector with oncolytic properties using a bispecific adapter protein

AUTHOR(S) : using a bispecific adapter protein  
Bian, Huijie; Wilden, Holger; Fournier, Philippe;

CORPORATE SOURCE: Peeters, Ben; Schirrmacher, Volker  
Division of Cellular Immunology, German Cancer  
Research Center, Heidelberg, D-69120, Germany

SOURCE: International Journal of Oncology (2006), 29(6), 1359-1369

CODEN: IJONES; ISSN: 1019-6439  
PUBLISHER: International Journal of Oncology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The aim of the study was: i) to specifically target tumor tissue by Newcastle disease virus (NDV) with oncolytic properties, ii) to improve the delivery system for systemic application of NDV via a bispecific adapter protein and iii) to investigate anti-tumor activity and side-effects. We selected two oncolytic virus strains, one native and the other recombinant, which showed multicyclic replication patterns in tumor cells. In order to reduce normal cell binding, they were modified by preincubation with a recombinant bispecific protein which blocks the viral native cell binding site and introduces a new binding site for a tumor-associated target (in this study, the interleukin-2-receptor, IL-2R). After i.v. transfer to mice, uptake of modified NDV in liver, spleen, kidney and lung was greatly reduced in comparison to unmodified NDV as determined by RRT-PCR of viral M gene copies. In IL-2R+ tumor bearing mice, the same assay revealed a high replication efficiency of the modified virus in the tumor tissue. Tumor therapy expts. showed that the side-effects induced by systemic application were greatly reduced by the adapter protein and that the anti-tumor effects were mostly undiminished. The demonstration of significant systemic anti-tumor activity of this viral vector suggests potential for augmentation by inclusion of one or more therapeutic genes.  
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2006:741365 CAPLUS  
DOCUMENT NUMBER: 145:206288  
TITLE: Newcastle disease virus exerts oncolysis by both intrinsic and extrinsic caspase-dependent pathways of cell death  
AUTHOR(S): Elankumaran, Subbiah; Rockemann, Daniel; Samal, Siba K.  
CORPORATE SOURCE: Virginia-Maryland Regional College of Veterinary Medicine, University of Maryland, College Park, MD, 20742, USA  
SOURCE: Journal of Virology (2006), 80(15), 7522-7534  
CODEN: JOVIAM; ISSN: 0022-538X  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Newcastle disease virus (NDV), an avian paramyxovirus, is tumor selective and intrinsically oncolytic. Here, the authors present evidence that genetically modified, recombinant NDV strains are cytotoxic to human tumor cell lines of ecto-, endo-, and mesodermal origin. They show that cytotoxicity against tumor cells is due to multiple caspase-dependent pathways, of apoptosis independent of interferon signaling competence. The signaling pathways of NDV-induced, cancer cell-selective apoptosis are not well understood. The authors demonstrate that NDV triggers apoptosis by activating the mitochondrial/intrinsic pathway and that it acts independently of the death receptor/extrinsic pathway. Caspase-8-methylated SH-SY5Y neuroblastoma cells are as sensitive to NDV as other caspase-8-competent cells. This demonstrates that NDV is likely to act primarily through the mitochondrial death pathway. NDV infection results in the loss of mitochondrial membrane potential and the subsequent release of the mitochondrial protein cytochrome c, but the second mitochondrion-derived activator of caspase (Smac/DIABLO) is not released. In addition, we describe early activation of caspase-9 and caspase-3. In contrast, cleavage of caspase-8, which is predominantly activated by the death receptor pathway, is a TNF-related, apoptosis-inducing ligand (TRAIL)-induced late event in NDV

-mediated apoptosis of tumor cells. The data, therefore, indicate that the death signal(s) generated by NDV in tumor cells ultimately converges at the mitochondria and that it acts independently of the death receptor pathway. The cytotoxicity studies demonstrate that recombinant NDV could be developed as a cancer virotherapy agent, either alone or in combination with therapeutic transgenes. The authors have also shown that trackable oncolytic NDV could be developed without any reduction in oncolytic efficacy.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2006:724439 CAPLUS  
DOCUMENT NUMBER: 145:448738  
TITLE: Study on the suppressing effect of NDV on cultured bladder carcinoma cells (BST739) in vitro  
AUTHOR(S): Tang, Xingsan; Ma, Yazhen  
CORPORATE SOURCE: Department of Biology, Xiaogan University, Xiaogan, 432000, Peop. Rep. China  
SOURCE: Shaanxi Yixue Zazhi (2005), 34(5), 521-524  
CODEN: SYZAEL; ISSN: 1000-7377  
PUBLISHER: Shaanxi Yixue Zazhi Bianjibu  
DOCUMENT TYPE: Journal  
LANGUAGE: Chinese

AB The suppressing effect of Newcastle disease virus (NDV) on human bladder carcinoma cells (BS739) in vitro was studied. Human bladder carcinoma cells (BST739) were used to observe the suppressing effect of NDV on them. The cell growth and proliferation were measured by MTT assay method. Cell apoptosis was evaluated by electron microscope and *in situ* TUNEL (TdT-mediated dUTP-X nick end labeling). The expression of cell apoptosis associated genes was examined by immunohistochem. staining. NDV had an effect on tumor cells and finally caused their apoptosis. The cell growth and proliferation were inhibited obviously, depending on time and concentration. The histol. and ultrastructural changes

were observed by electron microscope and microscope in treated group. The apoptosis index was 9.9% and 17.8% at 24 h and 48 h *in situ* TUNEL in treated group, resp. Human bladder carcinoma cell (BST739) apoptosis was induced and the protein expression of cancer gene bcl-2 was down regulated, while that of p53, bax, Fas, Fas-L genes and cell factor TGF- $\beta$ 1 were up regulated after NDV culture. NDV might be an effective oncolytic agent to bladder carcinoma cells (BST739). It has an obvious direct suppressing effect on tumor cells in vitro.

L3 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2006:545449 CAPLUS  
DOCUMENT NUMBER: 145:44711  
TITLE: Differentially regulated interferon response determines the outcome of Newcastle disease virus infection in normal and tumor cell lines  
AUTHOR(S): Krishnamurthy, Sateesh; Takimoto, Toru; Scroggs, Ruth Ann; Portner, Allen  
CORPORATE SOURCE: Division of Virology, Department of Infectious Diseases, St. Jude Children's Research Hospital, Memphis, TN, 38105-2794, USA  
SOURCE: Journal of Virology (2006), 80(11), 5145-5155  
CODEN: JOVIAM; ISSN: 0022-538X  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Newcastle disease virus (NDV) is a neg.-strand RNA virus with oncolytic activity against human tumors. Its effectiveness against tumors and safety in normal tissue have been demonstrated in

several clin. studies. Here we show that the spread of NDV infection is drastically different in normal cell lines than in tumor cell lines and that the two cell types respond differently to beta interferon (IFN- $\beta$ ) treatment. NDV rapidly replicated and killed HT-1080 human fibrosarcoma cells but spread poorly in CCD-1122Sk human skin fibroblast cells. Pretreatment with endogenous or exogenous IFN- $\beta$  completely inhibited NDV replication in normal cells but had little or no effect in tumor cells. Thus, the outcome of NDV infection appeared to depend on the response of uninfected cells to IFN- $\beta$ . To investigate their differences in IFN responsiveness, we analyzed and compared the expression and activation of components of the IFN signal transduction pathway in these two types of cells. The levels of phosphorylated STAT1 and STAT2 and that of the ISGF3 complex were markedly reduced in IFN- $\beta$ -treated tumor cells. Moreover, cDNA microarray anal. revealed significantly fewer IFN-regulated genes in the HT-1080 cells than in the CDD-1122Sk cells. This finding suggests that tumor cells demonstrate a less-than-optimum antiviral response because of a lesion in their IFN signal transduction pathway. The rapid spread of NDV in HT-1080 cells appears to be caused by their deficient expression of anti-NDV proteins upon exposure to IFN- $\beta$ .

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2006:186404 CAPLUS  
DOCUMENT NUMBER: 144:323988  
TITLE: Oncolytic viruses for the treatment of malignant glioma  
AUTHOR(S): Merrill, Melinda K.; Selznick, Lee A.; Gromeier, Matthias  
CORPORATE SOURCE: Department of Molecular Genetics & Microbiology, Duke University Medical Center, Durham, NC, 27710, USA  
SOURCE: Expert Opinion on Therapeutic Patents (2006), 16(3), 363-371  
CODEN: EOTPEG; ISSN: 1354-3776  
PUBLISHER: Ashley Publications Ltd.  
DOCUMENT TYPE: Journal; General Review  
LANGUAGE: English  
AB A review. Malignant glioma is the most common primary malignancy of the human CNS. Despite decades of research, the current therapeutic strategy consists of a multimodal regimen of surgery, chemotherapy, and radiation. This course of therapy yields a median survival after diagnosis of .apprx.1 yr. This dismal prognosis inspires the ongoing development of novel oncolytic agents targeting glioma, which include gene therapy, immunomodulatory therapy, and oncolytic viruses. Oncolytic viruses are defined by their ability to target, replicate in, and lyse tumor cells without critically damaging surrounding noncancerous tissue. Although some viruses are naturally oncolytic and tumor-selective, the advent of modern recombinant DNA technol. has allowed the engineering of addnl. viruses with improved therapeutic indexes. This technol. advance has enabled rapid growth in the field of viral therapy. Reovirus, Newcastle disease virus (NDV), measles virus, adenovirus, poliovirus, and herpes simplex virus 1 are in preclin. and clin. development for use as oncolytic agents against malignant glioma. This report focuses on the recent patent literature in the field of oncolytic viruses for the treatment of malignant glioma.

REFERENCE COUNT: 71 THERE ARE 71 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2006:39784 CAPLUS  
DOCUMENT NUMBER: 144:106246

TITLE: A tumor vaccine containing anti-CD3 and anti-CD28 bispecific antibodies triggers strong and durable antitumor activity in human lymphocytes

AUTHOR(S): Haas, Claudia; Lulei, Maria; Fournier, Philippe; Arnold, Annette; Schirrmacher, Volker

CORPORATE SOURCE: Division of Cellular Immunology, German Cancer Research Center, Heidelberg, Germany

SOURCE: International Journal of Cancer (2005), Volume Date 2006, 118(3), 658-667

CODEN: IJCNAW; ISSN: 0020-7136

PUBLISHER: Wiley-Liss, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We recently reported on newly designed virus-targeted bispecific CD3- and CD28-binding mols. for human T-cell activation. When bound via one arm to a human virus-modified tumor cell vaccine, these reagents caused a polyclonal T-cell response and overcame the potential various T-cell evasion mechanisms of tumor cells. In our current study, we demonstrated the induction of strong antitumor activity in human lymphocytes upon coincubation with a virus-modified tumor vaccine containing anti-CD3 and anti-CD28 bispecific antibodies. Blood mononuclear cells or purified T cells that were coincubated with such a tumor vaccine for 3 days were able to destroy monolayers of human breast carcinoma and other carcinoma cells. Serial transfer to new tumor cell monolayers revealed antitumor cytotoxic activity in such effector cells that lasted for about 10 days. Nontumor target cells appeared to be much less sensitive to the activated effector cells. Although the bispecific mols. alone did not activate effector cells, their binding to virus-infected tumor cells was important and more effective than their binding to free virus. Antitumor activity of the activated effector cells was mediated through soluble factors as well as through direct cell contact of effector cells with the nontargeted bystander tumor cells. Since the virus-modified tumor vaccine is well tolerated and already exhibits a certain effectiveness in cancer patients, the combination with new bispecific mols. has the potential to introduce addnl. antitumor effects. The reagents can also be combined with Newcastle Disease Virus (NDV)-based oncolytic virotherapy.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:1302066 CAPLUS

DOCUMENT NUMBER: 144:142318

TITLE: Phase I/II Trial of Intravenous NDV-HUJ Oncolytic Virus in Récurrent Glioblastoma Multiforme

AUTHOR(S): Freeman, Arnold I.; Zakay-Rones, Zichria; Gomori, John M.; Linetsky, Eduard; Rasooly, Linda; Greenbaum, Evgeniya; Rozenman-Yair, Shira; Panet, Amos; Libson, Eugene; Irving, Charles S.; Galun, Eithan; Siegal, Tali

CORPORATE SOURCE: Goldyne Savad Institute of Gene Therapy, Hadassah University Hospital, Jerusalem, 91120, Israel

SOURCE: Molecular Therapy (2006), 13(1), 221-228

CODEN: MTOHCK; ISSN: 1525-0016

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We undertook a Phase I/II trial in patients with apparent recurrent glioblastoma multiforme (GBM) based on imaging studies to determine the safety and tumor response of repetitive i.v. administration of NDV-HUJ, the oncolytic HUJ strain of Newcastle disease virus. The first part of the study utilized an accelerated intrapatient dose-escalation protocol with one-cycle dosage steps of 0.1, 0.32, 0.93, 5.9, and 11

billion infectious units (BIU) of NDV-HUJ (1 BIU = 1 + 10<sup>9</sup> EID50 50% egg infectious dose) followed by three cycles of 55 BIU. Virus was administered by i.v. infusion over 15 min. In the second part, patients received three cycles of 11 BIU. All patients without progressive disease were maintained with two doses of 11 BIU iv weekly. Eleven of the 14 enrolled patients (11-58 years, Karnofsky performance scale 50-90%) received treatment. Toxicity was minimal with Grade I/II constitutional fever being seen in 5 patients. Maximum tolerated dose was not achieved. Anti-NDV hemagglutinin antibodies appeared within 5-29 days. NDV-HUJ was recovered from blood, saliva, and urine samples and one tumor biopsy. One patient achieved a complete response. I.v. NDV-HUJ is well tolerated. The findings of good tolerability and encouraging responses warrant the continued evaluation of NDV-HUJ in GBM, as well as other cancers.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:151814 CAPLUS

DOCUMENT NUMBER: 143:110234

TITLE: Selective gene transfer to tumor cells by recombinant Newcastle disease virus via a bispecific fusion protein

AUTHOR(S): Bian, Huijie; Fournier, Philippe; Moormann, Rob; Peeters, Ben; Schirrmacher, Volker

CORPORATE SOURCE: Division of Cellular Immunology, German Cancer Research Center, Heidelberg, D-69120, Germany

SOURCE: International Journal of Oncology (2005), 26(2), 431-439

CODEN: IJONES; ISSN: 1019-6439  
PUBLISHER: International Journal of Oncology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Much interest exists presently in development of vectors for gene therapy of tumors based on RNA viruses because these viruses replicate in the cytoplasm and do not integrate into DNA. The neg. stranded paramyxovirus, Newcastle Disease Virus (NDV) from chicken has the addnl. advantages of preferential replication in tumor cells and of oncolytic and immunostimulatory properties. We here describe the bispecific fusion protein  $\alpha$ HN-IL-2 which binds to NDV, inhibits its normal cell binding property and introduces a new binding specificity for the interleukin-2 receptor (IL-2R). We demonstrate selective gene transfer to tumor cells expressing IL-2R via the bispecific fusion protein when using recombinant NDV carrying as marker gene the enhanced green fluorescence protein (NDFL-EGFP). Hemadsorption (HA) and neuraminidase activities (NA) of the HN protein of NDV were shown to be blocked by  $\alpha$ HN-IL-2 simultaneously and the absence of HA-activity of modified NDV was confirmed in vivo. Retargeted virus-binding to IL-2R pos. tumor cells was not sufficient for the process of cellular infection. It required in addition membrane fusion via the viral F-protein. By modification of recombinant NDV with a bispecific mol., our results demonstrate a novel and safe strategy for selective gene transfer to targeted tumor cells.

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:363266 CAPLUS

DOCUMENT NUMBER: 140:417458

TITLE: Syncytia Induction Enhances the Oncolytic Potential of Vesicular Stomatitis Virus in Virotherapy for Cancer

AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Kournioti, Chryssanthi; Park, Man-Seong; Garcia-Sastre, Adolfo;

CORPORATE SOURCE: Woo, Savio L. C.  
 Carl C. Icahn Center for Gene Therapy and Molecular  
 Medicine, Mount Sinai School of Medicine, New York,  
 NY, 10029, USA  
 SOURCE: Cancer Research (2004), 64(9), 3265-3270  
 CODEN: CNREAA; ISSN: 0008-5472  
 PUBLISHER: American Association for Cancer Research  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English  
 AB Vesicular stomatitis virus (VSV) selectively replicates in tumor but not in normal cells and is being developed as an oncolytic agent for cancer therapy. Here we report the construction of a recombinant VSV capable of inducing syncytia formation between tumor cells through membrane fusion at neutral pH, which led to enhanced oncolytic properties against multifocal hepatocellular carcinoma (HCC) in the livers of immunocompetent rats. Recombinant VSV vectors were constructed by insertion into their genome a transcription unit expressing a control or fusion protein derived from Newcastle disease virus. In vitro characterization of the recombinant fusogenic VSV vector on human and rat HCC cells showed extensive syncytia formation and significantly enhanced cytotoxic effects. In vivo, administration of fusogenic VSV into the hepatic artery of Buffalo rats bearing syngeneic multifocal HCC lesions in their livers resulted in syncytia formation exclusively within the tumors, and there was no collateral damage to the neighboring hepatic parenchyma. The fusogenic VSV also conferred a significant survival advantage over a nonfusogenic control virus in the treated animals ( $P = 0.0078$ , log-rank test). The results suggest that fusogenic VSV can be developed into an effective and safe therapeutic agent for cancer treatment in patients, including those with multifocal HCC in the liver.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 2003:221456 CAPLUS  
 DOCUMENT NUMBER: 138:251446  
 TITLE: Apathogenic strains of Newcastle Disease virus for treatment of cancer  
 INVENTOR(S): Zakay-Rones, Zichria; Panet, Amos; Irving, Charles  
 PATENT ASSIGNEE(S): Yissum Research Development Company, Israel; Ovcure Inc.  
 SOURCE: PCT Int. Appl., 51 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003022202	A2	20030320	WO 2002-IL765	20020912
WO 2003022202	A3	20040318		
WO 2003022202	A9	20040422		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2458543	A1	20030320	CA 2002-2458543	20020912
EP 1424897	A2	20040609	EP 2002-775172	20020912

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK  
 JP 2005508158 T 20050331 JP 2003-526332 20020912  
 US 2005031642 A1 20050210 US 2004-800256 20040311  
 PRIORITY APPLN. INFO.: IL 2001-145397 A 20010912  
 WO 2002-IL765 W 20020912

AB The present invention relates to lentogenic (apathogenic) strains of Newcastle Disease virus (NDV) that have oncolytic activities, and the use of such viruses and/or isolated proteins derived from all strains of the NDV virus in the treatment of cancer. The present invention thus provides compns. and methods for treatment of cancer using lentogenic oncolytic strain of nonhuman virus, the Newcastle Disease virus (NDV). It further provides methods for treatment of cancer comprising isolated viral proteins or subunits or analogs thereof having oncolytic activity as well as isolated polynucleotides or constructs containing same, which encode for the viral proteins. The polynucleotides or constructs containing same may include any vector polynucleotide, including viral vector polynucleotide. The present invention provides host cells containing the polynucleotides, constructs containing same, and the vector polynucleotides as described above, which will also be used for treatment of cancer. The present invention further provides treatment of cancer using combination of any of the above. A modified lentogenic NDV strain denoted herein as HUJ is disclosed below. The HUJ strain was compared to MTH-68/H strain of NDV, which is an attenuated strain obtained by serial passages through eggs (allantoic fluid), manufactured in Hungary by Phylaxia-Sanofi [Csatary and al. Anticancer Research (1999) 19-(1B):635-8]. The effect of MTH strain on cytotoxicity (Fig. 1) and apoptosis (Fig. 2) is more rapid than that observed with the HUJ strain. However, after 96 h of incubation both strains exhibit identical effect. Both viruses were also found to arrest cell replication. A rapid inhibition of DNA synthesis (90-95 %) was observed after 1 h of interaction of cells with NDV strains and fractions RO, RHN, B-1 and BHN.

L3 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 2002:68441 CAPLUS  
 DOCUMENT NUMBER: 137:134216  
 TITLE: Replication-competent, oncolytic Newcastle disease virus for cancer therapy  
 AUTHOR(S): Lorence, Robert M.; Roberts, M. Scot; Groene, William S.; Rabin, Harvey  
 CORPORATE SOURCE: Department of Viral Therapeutics, Pro-Virus, Inc., Gaithersburg, USA  
 SOURCE: Monographs in Virology (2001), 22 (Replication-Competent Viruses for Cancer Therapy), 160-182  
 CODEN: MONVAK; ISSN: 0077-0965  
 PUBLISHER: S. Karger AG  
 DOCUMENT TYPE: Journal; General Review  
 LANGUAGE: English

AB A review discusses the use of Newcastle disease virus (NDV) for cancer therapy. NDV has several properties that help differentiate it from other viruses for cancer therapy. Cytolytic strains of NDV have key features as replication-competent, oncolytic agents. Their high oncolytic potency and tumor selectivity are particularly important for systemic administration which is being explored in a current phase-I i.v. trial of advanced cancer patients using PV701, a cytolytic NDV strain.

REFERENCE COUNT: 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
 ACCESSION NUMBER: 2001:669671 CAPLUS  
 TITLE: Newcastle disease virus therapy of human tumor xenografts: antitumor effects of local or systemic

AUTHOR(S): administration  
Phuangsab, A.; Lorence, R. M.; Reichard, K. W.;  
Peeples, M. E.; Walter, R. J.  
CORPORATE SOURCE: Department of Surgery, Cook County Hospital, Chicago,  
IL, 60612, USA  
SOURCE: Cancer Letters (Shannon, Ireland) (2001), 172(1),  
27-36  
CODEN: CALEDQ; ISSN: 0304-3835  
PUBLISHER: Elsevier Science Ireland Ltd.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Previously we showed that a single local injection of the avian paramyxovirus Newcastle disease virus (NDV) strain 73-T caused long-lasting, complete tumor regression of human neuroblastoma and fibrosarcoma xenografts in athymic mice. Here we report the antitumor effects of NDV administered by either the intratumoral (IT) route to treat a variety of human carcinoma xenografts or by the systemic (i.p., IP) route to treat neuroblastoma xenografts (6.5-12 mm in diameter). For IT treatments, mice were randomized into treatment groups and given a single IT injection of NDV 73-T, vehicle (phosphate buffered saline, PBS), or UV-inactivated NDV. For systemic therapy, mice (n=18) with s.c. IMR-32 human neuroblastoma xenografts received IP injections of NDV (5+109 PFU). Significant tumor growth inhibition (77-96%) was seen for epidermoid (KB8-5-11), colon (SW620 and HT29), large cell lung (NCI-H460), breast (SKBR3), prostate (PC3), and low passage colon (MM17387) carcinoma xenografts treated IT with NDV. In all cases, tumors treated IT with PBS or replication-incompetent, UV-inactivated NDV displayed rapid tumor growth. After a single IP injection of NDV, complete regression of IMR-32 neuroblastomas was observed in 9 of 12 mice without recurrence for the 3-9 mo follow-up period. Six mice with recurrent neuroblastomas after one IP injection received one to three addnl. IP treatments with NDV. Three of these six mice showed complete regression without recurrence. These data show that: (1) NDV administered either IT or IP is an effective antitumor therapy in this system, (2) replication competency is necessary for maximal effect, and (3) multiple NDV doses can be more effective than a single dose. These studies provide further rationale for the preclin. study of NDV as an oncolytic agent.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2001:294496 CAPLUS  
DOCUMENT NUMBER: 135:342896  
TITLE: Induction of apoptosis by a Newcastle Disease Virus vaccine (MTH-68/H) in PC12 rat pheochromocytoma cells  
AUTHOR(S): Fabian, Zsolt; Torocsik, Beata; Kiss, Katalin; Csاتary, Laszlo K.; Bodey, Bela; Tigyi, Jozsef; Csاتary, Christine; Szeberenyi, Jozsef  
CORPORATE SOURCE: Department of Medical Biology, Pecs University, Pecs, Hung.  
SOURCE: Anticancer Research (2001), 21(1A), 125-135  
CODEN: ANTRD4; ISSN: 0250-7005  
PUBLISHER: International Institute of Anticancer Research  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The attenuated Newcastle Disease Virus (NDV) vaccine MTH-68/H has been found to cause regression of various tumors including certain types of human neoplasms. The mechanism of its oncolytic action is poorly understood, but it appears to affect specific signaling pathways in the target cell. We studied the cellular effects of NDV employing PC12 rat pheochromocytoma cells, a widely used model system to analyze differentiation, proliferation and apoptosis. The MTH-68/H

vaccine was found to be cytotoxic on PC12 cells. It caused internucleosomal DNA fragmentation, the most characteristic feature of programmed cell death (PCD). A brief exposure (30 min) of P12 cells to the virus was sufficient to produce a full-blown apoptotic response. Major mitogen-activated protein kinase pathways (including the stress inducible c-Jun N-terminal kinase pathway and p38 pathway) or mechanisms regulated by reactive oxygen species appear to have no role in virus-induced cell death. The PCD-inducing effect of MTH-68/H could not be prevented by simultaneous treatment of the P12 cells with growth factors or second messenger analogs stimulating protein kinase C or Ca<sup>++</sup>-mediated pathways. In contrast, treatment with a cAMP analog partially protected them from virus-induced apoptosis. These exptl. results suggests that MTH-68/H might disrupt a growth factor-stimulated survival pathway and that direct stimulation of protein kinase A-catalyzed phosphorylation events bypass this NDV-induced block.

REFERENCE COUNT: 110 THERE ARE 110 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 2000:85153 CAPLUS  
DOCUMENT NUMBER: 132:305651  
TITLE: Newcastle disease virus (NDV): brief history of its oncolytic strains  
AUTHOR(S): Sinkovics, J. G.; Horvath, J. C.  
CORPORATE SOURCE: St. Joseph's Hospital, Cancer Institute, Tampa, FL, USA  
SOURCE: Journal of Clinical Virology (2000), 16(1), 1-15  
CODEN: JCVIFB; ISSN: 1386-6532  
PUBLISHER: Elsevier Science Ireland Ltd.  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Background: While genetically engineered viruses are now being tested for the virus therapy of human cancers, some naturally occurring viruses display unmatched oncolytic activity. Newcastle disease virus (NDV) excels as an oncolytic agent. Objectives: As its virulence vs. attenuation can be explained on mol. biol. bases, it may be possible to develop or select highly oncolytic strains of NDV without adverse toxicity. Study design: Questions are posed as to the mechanisms of viral oncolysis, the appropriateness of tests to predict oncolytic activity of a given NDV strain and the best modes of administration for oncolytic effects. Answers are provided based on specific data or on considerations drawn from experience (the authors use NDV oncolyzates to immunize against melanoma and kidney carcinoma) or from analogous clin. situations (therapeutic use of mumps or measles viruses). Results and conclusions: NDV oncolyzates probably suit better for immunotherapy (providing also active tumor-specific immunization) than massive repeated inoculations of NDV strains, especially when the NDV strain used is not proven to be oncolytic by appropriate pre-clin. tests.

REFERENCE COUNT: 142 THERE ARE 142 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN  
ACCESSION NUMBER: 1988:628341 CAPLUS  
DOCUMENT NUMBER: 109:228341  
TITLE: Newcastle disease virus as an antineoplastic agent: induction of tumor necrosis factor- $\alpha$  and augmentation of its cytotoxicity  
AUTHOR(S): Lorence, Robert M.; Rood, Pamela A.; Kelley, Keith W.  
CORPORATE SOURCE: Dep. Anim. Sci., Univ. Illinois, Urbana, IL, USA  
SOURCE: Journal of the National Cancer Institute (1988),

80(16), 1305-12  
CODEN: JNCIEQ; ISSN: 0027-8874

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB The oncolytic strain 73-T of Newcastle disease virus (NDV) has been reported to be beneficial in the treatment of cancer patients, but little is known about its mechanism of action. NDV strain 73-T and a wild-type isolate of NDV were found to be potent inducers of tumor necrosis factor (TNF) production by both human peripheral blood mononuclear cells (PBMCs) and rat splenocytes. Antibody inhibition expts. identified TNF- $\alpha$  as the major species of TNF induced by NDV in PBMCs. Neither rHuTNF- $\alpha$  nor supernatants from NDV-stimulated PBMCs were cytotoxic toward the TNF-resistant human malignant melanoma cell line MEL-14. However, when MEL-14 cells were treated with NDV strain 73-T, both rHuTNF- $\alpha$  and supernatants from NDV-stimulated PBMCs killed 48% and 55%, resp., of these tumor cells. Treatment with NDV also conferred TNF susceptibility to the TNF-resistant human malignant melanoma cell line MEL-21 and the human myelogenous leukemia cell line K562. These results suggest two important mechanisms for the antineoplastic activity of NDV: (a) induction of TNF- $\alpha$  secretion by human PBMCs and (b) enhancement of the sensitivity of neoplastic cells to the cytolytic effects of TNF- $\alpha$ .

L3 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1970:130465 CAPLUS

DOCUMENT NUMBER: 72:130465

TITLE: Inhibitory effect of myxoviruses on a transplantable murine leukemia

AUTHOR(S): Eaton, M. D.; Scala, A. R.

CORPORATE SOURCE: Dep. of Bacteriol. and Immunol., Harvard Med. Sch., Boston, MA, USA

SOURCE: Proceedings of the Society for Experimental Biology and Medicine (1969), 132(1), 20-6

CODEN: PSEBAA; ISSN: 0037-9727

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Immunization of mice with parainfluenza viruses NDV or Sendai virus increases the oncolytic effect of these viruses when preinfected leukemic cells are injected into mice. Variations in oncolytic activity between different strains of influenza and parainfluenza viruses were noted, and also between 2 leukemic tumors induced by the Gross virus. Statolon given before virus-infected cells prevents oncolysis but has no effect when given later. Antiserum to NDV or Sendai (plus complement) shows a cytolytic effect in vitro against leukemic cells infected with these viruses.

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